

Applicants believe that the enclosed Declaration by the co-inventors demonstrates that the conception of the instant invention occurred as early as 1988, and that an actual reduction to practice occurred as early as September 13, 1993. The time period between November 16, 1992 and September 13, 1993 was consumed by the development of a knockout mouse model for atherosclerosis, and the testing of the mouse model to verify the inventive concept. As stated in the Declaration, the development of the mouse model began at least as early as 1989, and was completed on or about September 13, 1993. The mice were fed a high lipid diet for 8 months, commencing on or about September 13, 1993, and concluding on or about May 6, 1994, when the results of the experiment were collected and analyzed. The Examiner is urged to carefully consider the information presented in the Declaration.

Removal of Cummings et al. as a reference obviates the 35 U.S.C. 102(e) and 103(a) rejections.

As noted previously, applicants would be prepared to file a terminal disclaimer in this application to overcome the double patenting rejection assuming the application is otherwise considered to be in proper condition for allowance.

In view of the foregoing facts and reasons, the present application is now believed to overcome the remaining rejections, and to be in proper condition for allowance. Accordingly, reconsideration and withdrawal of the rejections, and favorable action on this application, is solicited. The Examiner is invited to contact the undersigned at the telephone number listed below to discuss the status of this application.

Respectfully submitted,

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MARKED-UP CLAIM

71. (Three Times Amended) A method for treating or inhibiting atherosclerosis in a mammal comprising:

providing an agent for inhibiting an interaction between P-selectin and PSGL-1 and between E-selectin and a ligand of E-selectin; and
administering said agent to a mammal in need of such treatment so as to cause such inhibition to occur, wherein said agent is selected from the group consisting of PSGL-1, soluble forms of PSGL-1, fragments of PSGL-1, and mimetics of PSGL-1, said agent being effective to inhibit the interaction between P-selectin and PSGL-1 and between E-selectin and a ligand of E-selectin.